



Stomach Cancer

Stomach cancer is relatively rare in the United States and most developed countries today. The incidence rate in Montana (5/100,000) is statistically significantly lower than the national incidence rate (8/100,000).¹ In Montana, stomach cancer accounts for only about 1% of all newly diagnosed cancers each year, although it is now the fourth most common incident cancer worldwide (down from the second most common in 2002), accounting for up to 20% of all cancers in some countries.²

The mucosa is the inner lining of the stomach. It is made up primarily of epithelial cells and contains glands that release digestive juices. More than 90% of stomach cancers start in the epithelial cells of the mucosa and penetrate into the submucosa layer and farther as they grow.

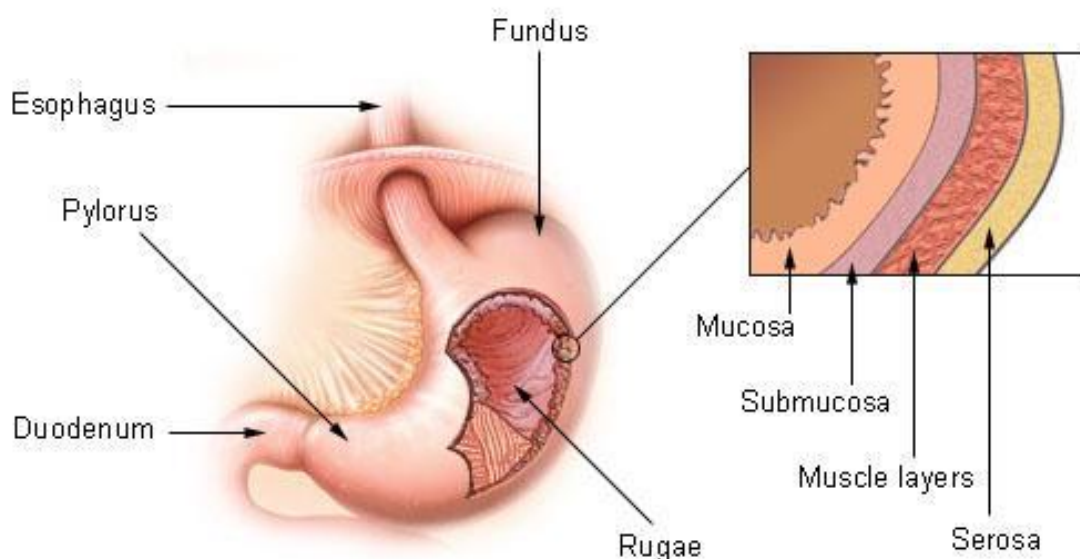
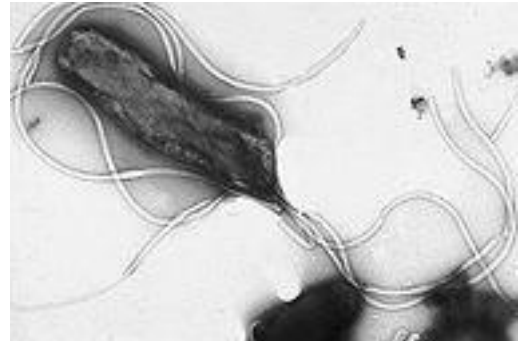


Image from the National Cancer Institute

¹ *Cancer in Montana 2003-2007*. Montana Central Tumor Registry Annual Report, November 2009.

² Parkin DM et al. 2005. Global cancer statistics, 2002. *CA: a Cancer Journal for Clinicians* **55** (2): 74-108.

The primary risk factors for stomach cancer are ulcers and atrophic gastritis, both secondary to colonization of the stomach lining by the bacterium *Helicobacter pylori*. To protect itself from the very acidic environment of the stomach, *H. pylori* invades the stomach lining, where it releases metabolic wastes that damage mucosal cells. This can cause chronic inflammation of the stomach lining, which can allow stomach acid to penetrate below the surface of the mucosa and damage deeper tissues, causing ulcers (erosions in the mucosa of the stomach or upper part of the small intestine caused by chronic irritation and inflammation) or chronic atrophic gastritis (destruction of gastric glands and permanent damage to the mucosa, which is replaced by scar tissue).³ Approximately half of all people who have *H. pylori* colonization develop atrophic gastritis. *H. pylori* colonization, even in the absence of ulcers or atrophic gastritis, is also a risk factor for stomach cancer. Between 60% and 80% of stomach cancer is attributable to long-term *H. pylori* colonization.^{2,4}



Photomicrograph of *Helicobacter pylori* from the
National Research Council of Canada Image Gallery

H. pylori colonization is a very strong risk factor but it is not sufficient by itself to cause stomach cancer, because only about 5% of colonized individuals develop stomach cancer.⁵ There appear to be substantial variations in the carcinogenic potential of different strains of *H. pylori* and there appear to be genetic differences among individuals in susceptibility to stomach cancer in the presence of *H. pylori*.⁶ *H. pylori* colonization may not even be necessary for the development of all cases of stomach cancer. Smoking increases the risk of stomach cancer, independent of *H. pylori* status.⁷ Diets low in fruits and vegetables, diets high in salt-preserved foods, and diets high in foods containing *N*-nitroso compounds such as smoked and grilled foods, all appear to increase the risk of stomach cancer.⁸

³ Kusters JG et al. 2006. Pathogenesis of *Helicobacter pylori* infection. *Clin Microbiol Rev* 19:449-490.

⁴ National Cancer Institute, www.cancer.gov

⁵ Shibata A, Parsonnet J. 2006. Stomach Cancer. In D Schottenfeld and JF Fraumeni Jr., eds. *Cancer Epidemiology and Prevention*, 3rd ed. NY: Oxford University Press, pp. 707-720.

⁶ Polk DB, Peek RM Jr. 2010. *Nat Rev Cancer* 10:403-414

⁷ Ladeiras-Lopes et al. 2008. *Cancer Causes Control* 19:689-701.

⁸ Liu C, Russell RM. 2008. *Nut Rev* 66:237-249.

H. pylori colonization occurs worldwide. It is more common in developing countries but it is not rare in the United States and other industrialized countries. The exact routes of colonization are still unknown, but *H. pylori* appears to be transmitted by close personal contact within households or institutional settings, and possibly by water and food.⁹ *H. pylori* is consistently associated with inadequate supplies of safe water and with poor hygiene practices. Nearly all studies have found that *H. pylori* is more common in populations of lower than of higher socioeconomic status, however defined.⁹

Colonization is established in early childhood in developing countries but later in adult life in industrialized countries. In the United States, colonization is uncommon among children but increases with age: the prevalence is about 10% between 18 and 30 years and increases to approximately 50% in adults age 60 and older.¹⁰ This suggests a cohort effect -- in the past, many people were colonized as children, but more recent generations experienced a lower risk of colonization. This has been attributed to generally improved hygiene, which may have reduced exposure to *H. pylori*; and to the widespread use of antibiotics in the United States over the past 75 years, which may have the incidental effect of eliminating some colonization.⁹

Given the reduced prevalence of *H. pylori* colonization in children in the United States, we would expect the incidence of stomach cancer to have declined over the past 75 years, and that has in fact happened. The incidence rate in the United States declined by nearly half between 1975 (12/100,000) and 2006 (7/100,000).⁴ Prior to the 1970s, before the widespread establishment of tumor registries that assess cancer incidence, estimates of cancer burden were limited to mortality data. As recently as 1940, stomach cancer was the leading cause of cancer death among men in the United States, and the third leading cause among women.⁴ Today, stomach cancer it is only the 19th in most common cause of cancer death in Montana.¹

The dramatic reduction of stomach cancer incidence and mortality in the United States in the past 75 years has been called an “unplanned triumph” in cancer control.¹¹ Unlike the similarly dramatic reduction of cervical cancer, which occurred after the adoption of routine Pap smear screening beginning in the 1950s, the reduction of stomach cancer happened without specific

⁹ Brown LM. 2000. *Helicobacter pylori*: Epidemiology and routes of transmission. *Epidemiol Rev* 22:283-297.

¹⁰ Everhart JE et al. 2000. *J Infect Dis* 181:1359-1363.

¹¹ Howson CP et al. 1986. The decline in gastric cancer: epidemiology of an unplanned triumph. *Epidemiol Rev* 8:1-27.

cancer control interventions. *H. pylori* was not isolated and described until the 1980s and its association with stomach cancer was discovered a few years later.⁴ In the absence of treatment for *H. pylori*, which is uncommon, colonization is usually permanent, but the decline in stomach cancer incidence has been occurring steadily since about 1925 in the United States. This, combined with the decline in age-specific prevalence of colonization in the United States, suggests that public health improvements in hygiene, the widespread use of refrigeration supplanting salt- and smoke-preservation of foods, reduction in food contamination, and greater availability of fresh fruits and vegetables may all be contributing factors, both by decreasing the risk of *H. pylori* colonization and by decreasing the prevalence of other risk factors for stomach cancer.

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